for systolic, diastolic and mean pressures within the lumen ($P < .001$). Sac wall pressures were significantly higher than luminal pressures for both high and low settings ($P < .001$).

**Comment:** Arterial pressure waves contribute to wall stress within an abdominal aortic aneurysm sac. These pressure waves can be modified by changes in the aortic aneurysm sac and the sac contents. This study, along with studies from the University of Pittsburgh and Dartmouth, suggest areas of increased stress on certain points of the aneurysm wall. At some point it may be possible to map stress points on the wall of an abdominal aortic aneurysm and to predict likelihood of rupture with greater certainty than is currently possible with measurement of aneurysm diameter alone.

**Hemodynamic effect of intermittent pneumatic compression of the leg after infrainguinal arterial bypass grafting**

Delis KT, Husmann MJ, Szendro G, et al. Br J Surg 2004;91:429–34.

**Conclusion:** Intermittent pneumatic compression after infrainguinal bypass grafting improves infrainguinal graft flow velocity.

**Summary:** This study examined the immediate effects of intermittent pneumatic compression (IPC) applied to the foot ($IPC_{foot}$), the calf ($IPC_{calf}$), and both simultaneously ($IPC_{foot+calf}$) on the hemodynamics of infrainguinal bypass grafts. There were 18 femoral popliteal and 18 femoral distal autogenous vein grafts included in the study. All limbs had a resting ankle-brachial pressure index of 0.9 or greater. Graft surveillance and measurement of graft hemodynamics were conducted at rest and within 5 seconds of IPC in each mode, using duplex imaging. Outcome measures included peak systolic velocity (PSV), mean velocity (MV), end diastolic velocity (EDV), pulsatility index (PI), and volume flow in the graft.

All modes of intermittent pneumatic compression enhanced PSV and volume flow in both femoral popliteal and femoral distal bypass grafts. $IPC_{foot+calf}$ was the most effective. $IPC_{foot+calf}$ enhanced median PSV, MV, and volume flow in femoral-popliteal grafts by 49%, 236%, and 182%, respectively. It attenuated PI by 61%. $IPC_{foot+calf}$ enhanced median PSV, MV, and median volume flow in femoral distal grafts by 53%, 179%, and 273%, respectively. PI was attenuated by 63%.

**Comment:** The article is interesting but of little practical significance. To achieve significant enhancement of graft flow with IPC, the limb must be dependent and therefore is more subject to postoperative swelling. It is also unclear whether these devices will increase postoperative discomfort in the operated extremity. Pedal devices might be contraindicated in patients with pedal gangrene and calf devices contraindicated in patients with distal calf wounds. Finally, it is unknown whether the short-term increase in flow velocities provided by these devices will result in long-term graft patency.

**Incidence of chronic thromboembolic pulmonary hypertension after pulmonary embolism**

Pengo B, Lensing WA, Prins MH, Thromboembolic Pulmonary Hypertension Study Group. New Engl J Med 2004;350:2257–64.

**Conclusion:** Chronic thromboembolic pulmonary hypertension (CTPH) is a relatively common long-term serious complication of acute pulmonary embolism.

**Summary:** The authors conducted a prospective, long-term study to assess the incidence of symptomatic CTPH in patients with an acute episode of pulmonary embolism (PE) but without prior venous thromboembolism (VTE). Patients who had persistent and unexplained dyspnea during follow-up underwent transthoracic echo and, when supportive findings were present, ventilation-perfusion lung scanning and pulmonary angiography. CTPH was diagnosed if systolic and mean pulmonary artery pressures exceeded 40 and 25 mm Hg, respectively; pulmonary capillary wedge pressure was normal; and there was angiographic evidence of pulmonary artery disease.

The authors identified 314 consecutive patients with acute PE. Eighty-one were excluded because of coexisting conditions potentially responsible for nonthromboembolic pulmonary hypertension. Overall, 223 patients were followed. Median follow-up was 94.3 months, and maximum follow-up was 10 years. There were no patients lost to follow-up.

Of the 223 patients, 32 (14.3%) had recurrent episodes of documented VTE. The incidence of recurrent VTE was 4.9% at 3 months, 8.0% at 1 year, 22.1% at 5 years, and 29.1% at 10 years. Of the 223 patients, 18 died as a direct consequence of the first PE, with 17 deaths occurring on the first day and 1 on the second day. In 7 of 223 patients, symptoms developed that proved to be due to CTPH and in only 2 cases were symptoms preceded by recurrent PE. The cumulative incidence of symptomatic CTPH was 1% at 6 months, 3.1% at 1 year and 3.8% at 2 years. No cases occurred after 2 years among patients with more than 2 years of follow-up. Risk of CTPH was increased by a previous PE (odds ratio [OR], 19.0), younger age (OR, 1.79/decade), a larger perfusion defect (OR, 2.22/decile decrement in perfusion), and idiopathic PE at presentation (OR, 5.7).

**Comment:** The article indicates that CTPH is much more common than previously suspected, with previous articles suggesting an incidence of 0.1% to 0.5% following acute nonfatal PE (New Engl J Med 2001;345: 1465–72). The disease however, is not inevitably associated with symptomatic VTE (New Engl J Med 350;22:2236–8) and does not share usual systemic and coagulation risk factors with VTE. The whitish-yellow fibrotic organized thromboembolism in a patient with CTPH strongly resembles chronic venous thrombosis of the extremities. The key to CTPH may lie in the resolution of the thrombotic process rather than the original embolus.

**Prevention of disabling and fatal strokes by successful carotid endarterectomy in patients without recent neurologic symptoms: Randomised controlled trial**

Halliday A, Mansfield A, Marro J, Asymptomatic Carotid Surgery Trial (ACST) Collaborative Group. Lancet 2004;363:1491–502.

**Conclusion:** In asymptomatic patients younger than 75 years of age with ≥70% carotid stenosis on ultrasound, immediate carotid endarterectomy (CEA) decreases the net 5-year stroke risk from about 12% to about 6% (including a 3% perioperative stroke and death rate), with half of the 5-year benefit involving fatal or disabling strokes.

**Summary:** From 1993 to 2003, 3120 asymptomatic patients with "substantial carotid narrowing" were randomized equally between immediate CEA (half receiving CEA by 1 month, 88% by 1 year) and indefinite deferral of CEA (only 4% per year receiving CEA). Follow-up was for up to 5 years (mean, 3.4 years). Assessment of carotid stenosis was by locally validated criteria, with results generally rounded to 70%, 80%, or 90%. One hundred twenty-six hospitals, primarily in Europe, participated in the study. Surgeons participating in the study were required to submit 50 CEAs and have a documented stroke and death rate of <6% at 30 days.

The risk of stroke or death within 30 days of CEA was 3.1% (95% CI, 2.3%-4.1%). Combining perioperative events and nonperioperative strokes, net 5-year risk in immediate CEA patients versus all allocated deferral patients was 6.4% versus 11.8% for all stroke (net gain, 5.4% [3.0–7.8]); $P < .0001$), 3.5% versus 6.1% for fatal or disabling stroke (net gain, 2.5% [0.8–4.3]); $P = .004$), and 2.1% versus 4.2% for fatal strokes(net gain, 2.1% [0.6–3.6]; $P = 0.006$). Benefits were significant for both men and women; for those with about 70%, 80%, and 90% carotid artery narrowing on ultrasound; and for those younger than 65 years of age and those 65–74 years of age.

**Comment:** This is a later and larger variant of the ACAS trial. The study indicates benefit for patients with asymptomatic carotid stenosis who undergo prophylactic CEA. However, the data also indicate that, with good medical care, patients with high-grade carotid stenosis have an annual stroke risk of only about 2% without CEA. In addition, morbidity and mortality of CEA must be low for benefit to be achieved. Like ACAS, the authors found no stratification of benefit of CEA in patients stratified for increasing carotid stenosis, perhaps reflecting the variability of ultrasound diagnosis. In contrast to ACAS, this study found benefit for CEA for asymptomatic carotid stenosis in women.

**Reduced neuropsychological test performance in asymptomatic carotid stenosis: The Tromso Study**

Mathiesen EB, Waterloo K, Joakimsen O, et al. Neurology 2004; 62:695–701.

**Conclusion:** Carotid stenosis is associated with poor neuropsychological performance.

**Summary:** The authors sought to assess the relationship between asymptomatic carotid stenosis, silent magnetic resonance imaging (MRI) lesions, and neuropsychological tests performance. The study consisted of 189 subjects with ultrasound-documented carotid stenosis and 221 control subjects without carotid stenosis. The patients were recruited from a population study. People with previous strokes were excluded. Study subjects were assessed with neuropsychologic tests that evaluated attention, psychomotor speed, memory, speed of information processing, motor functioning, intelligence, and depression. Subjects underwent sagittal T1-weighted and axial and coronal T2-weighted spin echo MRIs.

Patients with carotid stenosis were found to have lower performance on neuropsychologic tests with regard to attention, psychomotor speed, memory, and motor functioning. Results were independent of MRI lesions. Cortical infarcts and white matter hyperintensities imaged by MRI were equally distributed among people with and without carotid stenosis. Lacunar infarcts were more frequent in the patients with carotid stenosis ($P = .03$). No differences were found among patients with and without carotid stenosis with regard to speed of information processing, word association, or depression.

**Comment:** The impact of carotid stenosis on cognitive function continues to be debated. This article indicates that carotid stenosis is associated with adverse performance in some aspects of neuropsychologic testing. We do not know if it is the carotid stenosis itself or the conditions that lead to carotid stenosis that actually affect neuropsychologic testing. This study does not address whether correction of carotid stenosis results in improvement of neuropscyhologic testing or slows degeneration of the neuropsychologic functions potentially affected by with carotid stenosis.